

Climate, Traffic-Related Air Pollutants, and Asthma Prevalence in Middle-School Children in Taiwan

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This study compared the prevalence of asthma with climate and air pollutant data to determine the relationship between asthma prevalence and these factors. We conducted a nationwide survey of respiratory illness and symptoms in middle-school students in Taiwan. Lifetime prevalences of physician-diagnosed asthma and of typical symptoms of asthma were compared to air monitoring station data for temperature, relative humidity, sulfur dioxide, nitrogen oxides, ozone, carbon monoxide, and particulate matter with aerodynamic diameter $\leq 10 \mu\text{m}$ (PM_{10}). A total of 331,686 nonsmoking children attended schools located within 2 km of 55 stations. Asthma prevalence rates adjusted for age, history of atopic eczema, and parental education were associated with nonsummer (June–August) temperature, winter (January–March) humidity, and traffic-related air pollution, especially carbon monoxide and nitrogen oxides, for both girls and boys. Nonsummer temperature, winter humidity, and traffic-related air pollution, especially carbon monoxide and nitrogen oxides, were positively associated with the prevalence of asthma in middle-school students in Taiwan. **Key words:** air pollution, asthma, children, climate, environmental exposure. *Environ Health Perspect* 107:1001–1006 (1999). [Online 17 November 1999] <http://ehpnet1.niehs.nih.gov/docs/1999/107p1001-1006guo/abstract.html>

In humans, air pollutants are associated with mortality and morbidity secondary to respiratory diseases. In population studies, effects of air pollution have been associated with asthma morbidity and exacerbation of symptoms. Increased hospitalizations (1,2), emergency room and clinic visits (3,4), and medication use (5) due to asthma attacks, as well as decreased peak flow measurements (6) in asthmatics, have been associated with air pollution. However, the relationship between the prevalence of asthma and air pollution has yet to be proven. In Taipei, Taiwan, the increase in the prevalence of childhood asthma (from 1.3% in 1974 to 5.1% in 1985) among children 7–15 years of age has been attributed to deterioration in air quality (7). On the other hand, numerous studies in the United States and in Europe have shown no association between air pollution and reports of asthma (8,9). In clinical exposures of asthmatics, short exposure to air pollution produces increased airway reactivity. Thresholds in sulfur dioxide-induced bronchoconstriction were lower in asthmatics (10), and ozone-induced lung function change and respiratory tract injury and inflammation were more prominent in asthmatics (11).

Many factors contributed to the prevalence and/or attacks of asthma, including personal factors (smoking habits, genetics, age, sex, nutritional status, physiologic status, coexisting lung disease, lifestyle, allergy

status, family history, and occupation) and environmental stimuli (outdoor and indoor pollution, aeroallergens, climate) (12). The relative importance of outdoor air pollution exposure to the incidence and prevalence of asthma as compared to other intrinsic and environmental factors is not understood.

In this investigation, we compared asthma prevalence data from a nationwide survey of middle-school students with air pollutant and climatic data from 55 monitoring stations of the Taiwan Environmental Protection Administration (EPA; Taipei, Taiwan) to assess the effects of air pollutants on asthma.

Subjects and Methods

Respiratory health questionnaire. Between October 1995 and May 1996, we conducted a nationwide survey for respiratory diseases and symptoms in middle-school students. The study protocol was approved by the EPA Respiratory Health Screening Steering Committee and the Institution Review Board at National Cheng Kung University Medical College (Tainan, Taiwan), and complied with the principles outlined in the Helsinki declaration (13). Chinese versions of two questionnaires were used: the parent core questionnaire, which was taken home by the students, and the standard International Study of Asthma and Allergies in Childhood, Chinese version, a video questionnaire (14)

administered to students in schools. Classroom incentives but not individual incentives were used to encourage participation. The overall response rate was 89%, with a range of 87–93% for the individual schools.

Definition of diseases and symptoms.

Two indicators of asthma were considered. Physician-diagnosed asthma was defined by parental reports of the child ever being diagnosed by a physician as having asthma. The video questionnaire determined asthmatic symptoms including dyspnea, wheezing, exercise-induced wheezing, wheezing, or dry cough at night unassociated with a cold. Questionnaire-determined asthma was defined by the report of dyspnea and nocturnal dyspnea associated with wheezing (from the video questionnaire), the report of attacks of dyspnea with wheezing (from the parental questionnaire), or physician-diagnosed asthma (reported by a parent). Atopic eczema was defined as the presence of itching skin eruption at cubital, posterior popliteal, neck, periauricle, and eyebrow areas for 6 months or longer, and a diagnosis of atopic eczema by a physician. Information on lifetime prevalences of asthma and atopic eczema was obtained using these definitions.

Air pollutant monitoring data.

Complete monitoring data for the air pollutants SO_2 , nitrogen oxides (NO_x), O_3 , carbon monoxide, and particles with aerodynamic diameter $\leq 10 \mu\text{m}$ (PM_{10}) were available from 66 EPA monitoring stations in 1994 and later years. Concentrations of each pollutant were measured continuously and reported hourly—CO by nondispersive

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infrared absorption, NO_x by chemiluminescence, O_3 by ultraviolet absorption, SO_2 by ultraviolet fluorescence, and PM_{10} by beta-gauge. Temperature and relative humidity were measured daily at each station. The study population was limited to children attending schools located within 2 km of 55 of these monitoring stations (Figure 1).

Site-specific average annual air pollution, temperature, and humidity were calculated as the mean of the 1994 monthly averages. Temperature and relative humidity were highly correlated across the sites. Principal component factor analysis with varimax rotation (15) was used to produce independent indicators of the site-specific climate. There were also strong between-site correlations of the five monitored air pollutants SO_2 , NO_x , O_3 , CO, and PM_{10} . Principal component factor analysis with varimax rotation was also used to define source-specific indicators of air pollution exposure.

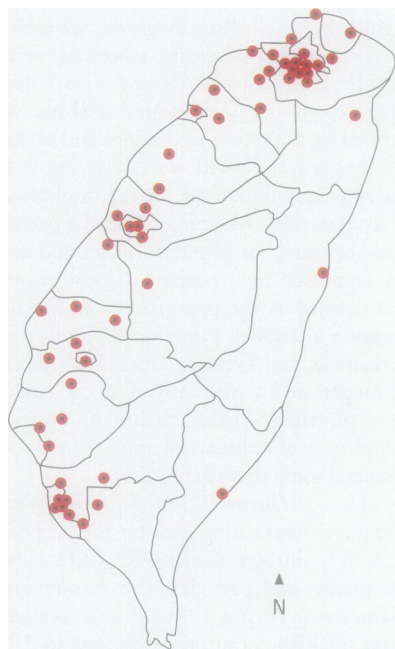


Figure 1. The 55 air pollution monitoring stations in this study and their 2-km catchment areas in Taiwan, 1994.

Statistical analysis for health and exposure associations. Asthma is associated with individual factors such as age, sex, atopy, and socioeconomic status. We used two-stage methods to analyze the prevalence of asthma in each gender. In the analysis of both physician-diagnosed and questionnaire-determined asthma rates, logistic regression was used to estimate the adjusted illness frequency in each of the 55 catchment areas, controlling for age, history of atopic eczema, and parental education. The sex-specific prevalence rate of asthma among students was calculated for each catchment area. Self-reported active smokers were excluded. In the second step, these site-specific adjusted asthma prevalence rates were regressed against the area-specific temperature, relative humidity, and air pollution factors using weights proportional to the inverse variance ($1/\text{standard error}^2$) of the adjusted prevalence rates. The adjusted prevalence rates were plotted against the scores of air pollutant principal components after adjusting for the temperature and humidity variables.

Results

In September 1995 there were 1,139,452 students enrolled in 800 middle schools in

24 counties and cities in Taiwan. A total of 1,018,031 (89.3%) students and their parents responded satisfactorily to the questionnaire. The prevalence of asthma ranged from 4.6% in Yunlin County, central Taiwan, to 12.0% in Taipei City, northern Taiwan. Among these students, 161,744 boys and 170,942 girls were nonsmokers and were enrolled in a school within 2 km of an air pollutant monitoring station. Table 1 shows the characteristics of study subjects by sex. Figure 2 summarizes the 1994 annual mean concentration of criteria air pollutants, the temperature, and the relative humidity in the 55 monitoring stations. Table 2 summarizes the site-specific air pollution and meteorologic annual means at these sites.

Two factors were identified from the principal component analysis of the site-specific temperature: summer and nonsummer month temperatures (Table 3). Three factors were found for relative humidity: spring dryness, winter humidity, and the second-half year humidity (Table 4). Two factors were identified for the site-specific air pollutants (Table 5). The first site-specific factor was positively associated with CO and NO_x and negatively associated with O_3 , suggesting motor vehicle emissions; the second was

Table 1. Prevalence rate of asthma by characteristics of the study population in Taiwan as of 1 January 1996.

	Boys			Girls		
	Number (%)	Physician-diagnosed asthma (%)	Questionnaire-determined asthma (%)	Number (%)	Physician-diagnosed asthma (%)	Questionnaire-determined asthma (%)
Age (years)						
<13	36,202 (22.7)	7.1	12.5	37,297 (22.3)	4.3	8.3
13	54,661 (34.3)	6.5	11.1	56,816 (33.9)	4.2	8.0
14	53,618 (33.7)	5.9	10.1	57,240 (34.2)	3.7	7.1
≥ 15	14,650 (9.2)	5.4	9.2	16,078 (9.6)	3.8	7.3
Education level (years) ^a						
< 6	29,540 (18.6)	4.2	8.4	32,470 (19.4)	2.6	6.3
6–8	39,781 (25.0)	4.7	9.2	41,177 (24.6)	3.1	6.5
≥ 9	89,810 (56.4)	7.8	12.5	93,784 (56.0)	4.9	8.7
Atopic eczema						
No	157,418 (98.9)	6.2	10.7	165,739 (99.0)	3.9	7.5
Yes	1,713 (1.1)	21.3	30.1	1,692 (1.0)	17.1	23.8

Total boys, $n = 159,131$; total girls, $n = 167,431$.

^aParents.

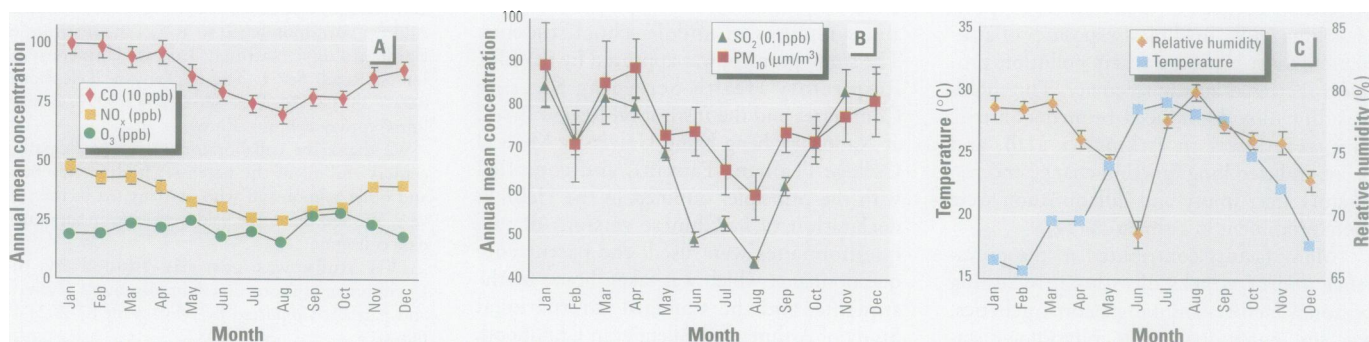


Figure 2. Summary of area-specific annual mean air pollutant concentrations: 55 monitoring stations in Taiwan, 1994. (A) Traffic-related air pollutants. (B) Nontraffic-related air pollutants. (C) Temperature and relative humidity. Error bars indicate standard error.

highly associated with SO₂ and PM₁₀, suggesting emissions from power plants, industry, and domestic fossil fuel combustion, as recently reviewed in Taiwan (16).

Physician-diagnosed (Table 6) and questionnaire-determined (Table 7) asthma was regressed against the two temperature factors, three relative humidity factors, and two air pollutant factors. Physician-diagnosed asthma was more prevalent in communities with higher temperature in nonsummer months and higher traffic-related air pollution for both girls and boys (Table 6). Questionnaire-determined asthma was more prevalent in communities with higher temperature in nonsummer months, higher humidity in winter months, and higher traffic-related air pollution for both girls and boys (Table 7).

Adjusted site-specific asthma prevalence was plotted against the traffic-related air pollution factor scores (Figures 3 and 4). No outlier effects were apparent, and the regression line appeared linear.

The effect of each pollutant on the prevalence rate of physician-reported and questionnaire-determined asthma was assessed separately and expressed as the difference in prevalence rate across interquartile range for each air pollutant (Table 8). Positive statistically significant associations

were found for CO and NO_x; negative or very weak associations were found for O₃, SO₂, and PM₁₀. Although boys had higher lifetime prevalence of asthma than girls in all levels of air pollution, there were no statistically significant differences in effect estimates for boys versus girls, indicating similar effects of air pollutants on asthma prevalence in both sexes.

Discussion

This study is based on a nationwide survey of asthma linked to a local air pollution measurement. The prevalence of physician-diagnosed and questionnaire-determined asthma of middle-school students from 55 locations was associated with temperature, humidity, and traffic-related air pollutants, especially CO and NO_x.

The climatic factors, especially the nonsummer temperature and winter humidity, are associated with the lifetime cumulative incidence of asthma. This is compatible with findings in New Zealand, where an increase of 1°C in annual average temperature is associated with a 1% increase of long-term asthma prevalence, but not an acute exacerbation of asthma (17). Exposure to house dust mite allergen is the most important factor in asthma in Taiwan (18). Increased temperature

(up to 30°C) and increased relative humidity favor the development of mites (19). This is especially true during the colder seasons. Although in areas with lower humidity indoor dampness is probably more important than outdoor humidity in the domestic development of mites (20), the high outdoor humidity in Taiwan (monthly average 68–80%; Figure 2C) could have been an important determinant of the indoor humidity. The results of this survey suggest that exposure to dust mites or other humidity sensitive allergens was elevated in the children from warm and humid areas in Taiwan.

Because of the close correlation among the air pollutants, it is impossible to separate the effects of individual pollutants. Therefore, we used factor analysis to group the patterns of pollutants into two classes of pollutants: traffic-related and stationary fossil fuel-related (Table 4). We found a strong correlation between traffic-related pollutants and lifetime prevalence of asthma in both boys and girls (Figures 3 and 4). When the effect of each pollutant on the asthma prevalence rates was examined (Table 8), significant association was found for CO and NO_x. No consistent relationship was seen among asthma prevalence and O₃, SO₂, and PM₁₀. Whether air pollution caused increased prevalence of asthma has been a subject of long debate. High levels of sulfur dioxide and black smoke in East Germany were associated with lower prevalence of asthma as compared to West Germany (21). However, these data could not exclude a role for motor vehicle pollutants in increasing the occurrence of asthma. Children living near roads are more likely to have recurrent wheezing and dyspnea as well as reduced peak expiratory flow rate (22). Japanese women living near roadways are more likely to have chronic wheezing adjusted by age, smoking status, years of residence, occupation, and type of home heating (23). In Austria, lifetime prevalence of asthma was associated with long-term

Table 2. Mean and distribution of 1994 annual air pollution and meteorology data from 55 monitoring stations in Taiwan.

	Mean ± SD	Minimum	25th percentile	Median	75th percentile	Maximum
CO (ppb)	853 ± 277	381	675	843	1,001	1,610
NO _x (ppb)	35.1 ± 13.4	10.2	25.6	34.0	42.9	72.4
SO ₂ (ppb)	7.57 ± 4.15	0.88	5.01	7.22	8.77	21.2
PM ₁₀ (µg/m ³)	69.2 ± 17.8	40.1	54.0	65.9	81.7	116.2
O ₃ (ppb)	21.3 ± 4.5	12.4	18.7	21.5	23.4	34.1
Temperature (°C)	22.9 ± 1.1	19.6	22.3	22.8	23.6	25.1
Relative humidity	76.2 ± 3.7%	64.8%	74.8%	76.6%	78.6%	86.2%

Abbreviations: PM₁₀, particulate matter ≤ 10 µm in aerodynamic diameter; SD, standard deviation.

Table 3. Factor loading of the monthly average temperature in Taiwan.

	Factor 1	Factor 2
Eigenvalues ^a	9.27 (77.3%)	1.67 (14.0%)
January	<i>0.927</i>	0.303
February	<i>0.929</i>	0.283
March	<i>0.966</i>	0.188
April	<i>0.896</i>	0.235
May	<i>0.891</i>	0.309
June	0.398	<i>0.856</i>
July	0.338	<i>0.900</i>
August	0.082	<i>0.901</i>
September	<i>0.523</i>	<i>0.804</i>
October	<i>0.747</i>	<i>0.587</i>
November	<i>0.880</i>	0.434
December	<i>0.867</i>	0.413
Factor description	Nonsummer warmth	Summer warmth

Two factors have eigenvalues > 1 and account for 91.2% of variance. Values in italics are those rotated factor scores with absolute value > 0.5.

^aPercent variance explained.

Table 4. Factor loading of the monthly average of relative humidity in Taiwan.

	Factor 1	Factor 2	Factor 3
Eigenvalues ^a	5.62 (46.8%)	2.14 (17.8%)	1.35 (11.2%)
January	0.162	-0.016	<i>0.903</i>
February	0.160	-0.242	<i>0.902</i>
March	0.174	<i>-0.566</i>	<i>0.704</i>
April	0.122	<i>-0.809</i>	0.188
May	0.063	<i>-0.909</i>	0.139
June	0.209	<i>-0.737</i>	0.077
July	<i>0.745</i>	-0.331	0.200
August	<i>0.746</i>	-0.366	0.076
September	<i>0.902</i>	-0.239	0.095
October	<i>0.875</i>	-0.122	0.134
November	<i>0.804</i>	-0.048	0.206
December	<i>0.733</i>	0.110	0.046
Factor description	Summer and fall humidity	Spring dryness	Winter humidity

Three factors have eigenvalues > 1 and account for 75.9% of variance. Values in italics are those rotated factor scores with absolute value > 0.5.

^aPercent variance explained.

Table 5. Factor loading of the monthly average levels of criteria air pollutants in Taiwan.

	Factor 1	Factor 2
Eigenvalues ^a	2.92 (58.3%)	1.31 (26.2%)
Average CO level	<i>0.927</i>	0.127
Average NO _x level	<i>0.911</i>	0.278
Average O ₃ level	<i>-0.921</i>	0.038
Average SO ₂ level	0.268	<i>0.827</i>
Average PM ₁₀ level	-0.037	<i>0.901</i>
Factor description	Traffic-related	Stationary fossil fuel combustion-related

PM₁₀, particulate matter ≤ 10 µm in aerodynamic diameter. Two factors have eigenvalues > 1 and account for 84.5% of variance. Values in italics are those rotated factor scores with absolute value > 0.5.

^aPercent variance explained.

traffic nitrogen dioxide exposure in a dose-related fashion (24). In a Canadian case-control study to compare newly diagnosed asthmatic children and controls, NO₂ exposure of > 15 ppb by personal badge for 24 hr was associated with an odds ratio of 10 after adjusting for other risk factors, although the NO_x exposure included both outdoor and indoor sources (25). Our study findings are compatible with the hypothesis that exposure to traffic-related pollutants might have caused changes in the susceptibility of children to allergens and therefore may have contributed to the development of asthma.

Whether this was directly related to CO or NO_x from automobile exhaust or was actually caused by other traffic-related pollutants not measured by the monitoring station could not be determined in this study. It is unlikely that CO is causally related to asthma prevalence. CO affects cardiovascular function, although it has not been shown to be a respiratory irritant. NO_x are acute respiratory irritants in animal and controlled human exposure studies. However, in epidemiologic studies of indoor NO₂ exposures from gas

stoves, there is not strong or consistent evidence of associations with asthma prevalence.

High traffic density is inversely correlated with concentrations of O₃ (22), which is formed at some distance from emission sources and scavenged in city centers by nitrogen monoxide from vehicle exhaust. In our factor analysis, O₃ had a negative association with traffic-related pollutants (Table 5). From the individual pollutant regression analysis, O₃ had either negative or no association with lifetime prevalence of asthma (Table 8). This is compatible with the hypothesis that the direct emissions of motor vehicles, which scavenge O₃ and therefore are negatively correlated with O₃, are more important determinants of asthma prevalence than the secondary pollutants, such as O₃, that are formed downwind.

Other studies have suggested increased asthma prevalence associated with traffic emissions. Wjst and colleagues (26) found that school-specific prevalences of recurrent wheezing and dyspnea were positively associated with traffic volume in the school district. Edwards et al. (27) reported that hospital

admission of children younger than 5 years of age for asthma increased with traffic volume and decreased with distance from the nearest main road. Brunekreef et al. (28) found that lung function of children attending schools within 300 m of a major road decreased in association with truck-traffic volume, but not with automobile traffic.

CO and NO_x are important constituents of motor-vehicle emissions. Other major components of motor-vehicle exhausts not measured in this study include fine and ultra-fine particles (including various heavy metals) and a wide range of organic compounds. The PM₁₀ measurements were only weakly correlated across sites with traffic-related pollution, and likely reflect emission from sources other than motor vehicles. Fine particle concentrations were not associated with asthma prevalence in children in the 24 cities study, but this study specifically excluded communities with substantial traffic-related pollution (9). There also are few epidemiologic data on asthma associations with organic compounds. However, Ware et al. (29) reported that school-specific asthma prevalence in Kanawha

Table 6. Relationship between adjusted parent-reported physician-diagnosed asthma prevalence (%) and environmental factors in middle-school students.

	Boys ($R^2 = 0.57$)		Girls ($R^2 = 0.50$)	
	Estimate	95% confidence interval	Estimate	95% confidence interval
Intercept	5.8 [#]	(5.5, 6.2)	≥ 3.5 [#]	(3.3, 3.8)
Temperature				
Nonsummer	0.90 [#]	(0.41, 1.4)	0.59**	(0.20, 0.98)
Summer	0.59*	(0.12, 1.1)	0.41*	(0.059, 0.76)
Humidity				
Summer and fall	0.21	(-0.055, 0.47)	0.20	(-0.0024, 0.42)
Spring	-0.29	(-0.61, 0.042)	-0.23	(-0.49, 0.037)
Winter	0.37*	(0.0078, 0.73)	0.15	(-0.14, 0.43)
Air pollution				
Traffic-related	1.1 [#]	(0.67, 1.6)	0.69 [#]	(0.32, 1.06)
Stationary fossil fuel combustion-related	-0.58**	(-1.00, -0.17)	-0.032	(-0.68, 0.037)

Results were obtained by multiple regression using prevalence of physician-diagnosed asthma as the dependent variable and factor scores of temperature, relative humidity, and air pollutants as the independent variables.

* $p < 0.05$. ** $p < 0.01$. [#] $p < 0.001$.

Table 7. Relationship between adjusted questionnaire-determined asthma prevalence (%) and environmental factors in middle-school students.

	Boys ($R^2 = 0.51$)		Girls ($R^2 = 0.41$)	
	Estimate	95% confidence interval	Estimate	95% confidence interval
Intercept	10.3 [#]	(9.8, 10.7)	7.1 [#]	(6.7, 7.5)
Temperature				
Nonsummer	1.3 [#]	(0.66, 1.9)	1.10 [#]	(0.54, 1.7)
Summer	0.66*	(0.05, 1.27)	0.27*	(-0.25, 0.78)
Humidity				
Summer and fall	0.16	(-0.18, 0.50)	0.27	(-0.039, 0.58)
Spring	-0.38	(-0.80, 0.05)	-0.20	(-0.57, 0.18)
Winter	0.74**	(0.27, 1.21)	0.47*	(0.05, 0.89)
Air pollution				
Traffic-related	0.99**	(0.37, 1.60)	0.85**	(0.33, 1.37)
Stationary fossil fuel combustion-related	-0.13	(-0.67, 0.41)	-0.23	(-0.74, 0.28)

Results were obtained by multiple regression using prevalence of questionnaire-determined asthma as the dependent variable and factor scores of temperature, relative humidity, and air pollutants as the independent variables.

* $p < 0.05$. ** $p < 0.01$. [#] $p < 0.001$.

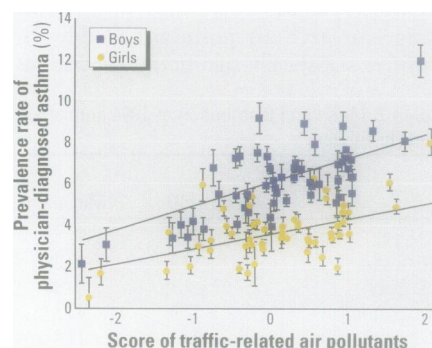


Figure 3. Association of physician-diagnosed asthma prevalence with traffic-related air pollutants adjusted for temperature and relative humidity in the 55 monitoring stations. Means and standard errors are shown.

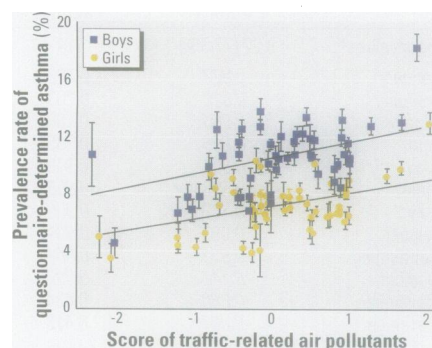


Figure 4. Association of questionnaire-determined asthma prevalence with traffic-related air pollutants adjusted for temperature and relative humidity in the 55 monitoring stations. Means and standard errors are shown.

County, West Virginia, increased with petroleum air pollution concentrations. In this study of asthma prevalence in Taiwan, CO and NO_x may have served as indicators of motor vehicle emissions rather than as direct measurements of the causal agent.

We found no association between lifetime asthma prevalence and nontraffic-related air pollutants, namely, SO₂ and PM₁₀. This is compatible with results from the Six Cities Study of Air Pollution and Health (30), which showed no correlation among PM₁₅, PM_{2.5}, and SO₂ and doctor-diagnosed asthma.

Questionnaires have been used to assess the prevalence of asthma in previous studies (31,32). Asthma is usually defined as current asthma (i.e., wheezing within the past 12 months). We used lifetime prevalence of asthma as one of our outcome variables to examine the effects of air pollution to the development of asthma. Underestimation of the true asthma prevalence is likely if parent reports of physician-diagnosed asthma are used. We compensated for this underestimation by adding self- and parent-reported wheezing, dyspnea, and nocturnal dyspnea, which we called questionnaire-determined asthma. In the 55 geographic areas studied, questionnaire-determined asthma had a strong correlation with physician-diagnosed asthma, with R² 0.79 for boys and 0.81 for girls.

There are advantages in the exposure information in this study. In Taiwan, most of the elementary and middle school students live close to their schools. Monitoring stations located near the schools are also likely to be near the students' homes, thus providing good surrogates for both school and home exposure. Heating and air conditioning are rare in Taiwanese classrooms. Air conditioning has become more popular for residential homes in recent years; heating is uncommon. Coal stoves were used in < 2% of homes in 1994, probably more in rural than in urban

areas (33). Not adjusting for coal stove exposure could have reduced the magnitude of association. The exposure information obtained from air pollution monitoring stations was limited to the criteria air pollutants in 1994. Air pollution measurements prior to 1994 were not available. Moving from one catchment area to another could have led to misclassification of exposure. However, the errors in exposure assessment are likely to be random, which would reduce the magnitude of association but would not introduce a positive bias in the associations.

The lack of personal exposure data prohibited analysis comparing individual exposure and outcomes. Therefore, the adjusted prevalence rate of asthma for each catchment area was used to determine the effects of air pollutants on the respiratory system. Because asthma was potentially associated with age, atopy, and socioeconomic status, these factors were controlled by the first-stage regression. Maternal smoking during pregnancy was associated with increased cord blood immunoglobulin E (34) and therefore might be a predictive factor of asthma. However, < 5% of Taiwanese women smoked (35), and obtaining maternal smoking history was considered somewhat sensitive. Therefore, maternal smoking history during pregnancy was not included in the questionnaire. There is no reason to believe that traffic-related pollutants were associated with maternal smoking. Smoking prevalence in women is lower in urban than in rural areas; therefore, if there is an adverse effect of maternal smoking during pregnancy on childhood asthma, it should have reduced the association between traffic-related pollutants and the prevalence of asthma. Secondary smoking can be a risk factor for asthma (36). Having an asthmatic child at home is a strong motivation for reduction or cessation of smoking. Because the causal relationship between secondary smoking and asthma in

children could not be determined for this cross-sectional design, we did not include passive smoking in our adjusting factors.

The studied population was a sample of the total population of middle-school children in Taiwan. Because information on air pollution is a prerequisite of this investigation, only the children in areas around air monitoring stations were chosen. The previous selection of sites by the EPA for establishing monitoring stations was nonrandom and probably favored places with expected high air pollution concentrations. As compared to the total number of middle-school students in Taiwan, selected subjects were similar in sex (male 50.4%) and age (average 13.8 years). However, the parents of these students had more education, smoked less, and complained more about air pollution problems.

In conclusion, parental reports of physician-diagnosed asthma and questionnaire-determined asthma are both associated with natural environmental factors such as temperature and humidity, probably mediated by exposure to common allergens such as dust mites. Asthma prevalence rates were highly correlated with traffic-related air pollutants (CO and NO_x) but not other pollutants (PM₁₀, O₃, and SO₂). Whether this is related to urban lifestyle, to exposure to CO and NO_x, or to other traffic-related pollutants warrants further investigation.

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Table 8. Effect of each pollutant on the prevalence rate of physician-diagnosed and questionnaire-determined asthma.

	Physician-diagnosed asthma		Questionnaire-determined asthma	
	Estimate (%)	95% confidence interval	Estimate (%)	95% confidence interval
Boys				
CO (ppb)	1.17	(0.63 - 1.72)	1.10	(0.45 - 1.75)
NO _x (ppb)	0.88	(0.23 - 1.52)	0.99	(0.28 - 1.70)
O ₃ (ppb)	-0.89	(-1.51 - -0.27)	-0.68	(-1.40 - -0.05)
SO ₂ (ppb)	-0.15	(-0.55 - 0.25)	0.11	(-0.34 - 0.56)
PM ₁₀ (μg/m ³)	-0.96	(-1.82 - -0.09)	-0.29	(-1.32 - 0.75)
Girls				
CO (ppb)	0.84	(0.45 - 1.22)	1.00	(0.44 - 1.56)
NO _x (ppb)	0.50	(0.03 - 0.97)	0.77	(0.13 - 1.42)
O ₃ (ppb)	-0.43	(-0.89 - 0.03)	-0.62	(-1.25 - -0.007)
SO ₂ (ppb)	0.027	(-0.28 - 0.33)	0.12	(-0.30 - 0.53)
PM ₁₀ (μg/m ³)	-0.52	(-1.18 - 0.13)	-0.41	(-1.33 - 0.52)

PM₁₀, particulate matter ≤ 10 μm in aerodynamic diameter. Effect is expressed as the difference in prevalence rate across interquartile range.

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